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CLAIMS

- 5 1. Use of peptide antagonists at glutamate receptors for the manufacture of a medicament to influence the glutamate-receptor-controlled cells.
- 2. Use of peptide antagonists at NMDA receptors for the manufacture of a medicament to influence the NMDA-receptor-controlled cells.
- 3. Use according to claim 2 in which the medicament prevents NMDA-receptor-mediated excitatory effects such as release of neurotransmitter or peptide as well as toxic effects resulting in cell injury or death.
 - 4. Use according to any of claims 1 to 3 in which the cells are neurons or glial cells in the central nervous system.
 - 5. Use according to any of claims 1 to 4 in which the medicament comprises glutamic acid-terminating peptides.
- 6. Use according to any of claims 1 to 5 in which the antagonist is chosen among (1-5)GnRH, (1-3)IGF-I, (1-37)GRF and C-peptide of insulin.
 - 7. Use according to any of claims 1 to 6 in which the medicament influence GnRH secretion.
 - 8. Use according to any of claims 1 to 7 for the treatment of acute or chronic disorders of the central nervous system.
- 9. Use according to any of claims 1 to 7 for the treatment of hypoxic, ischemic and metabolic brain disorders such as stroke and hypoglycaemia, traumatic, radiation-induced or inflammatory injuries to the brain and chronic degenerative states.

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- 10. Use according to any of claims 1 to 9 for the treatment of children during the perinatal period and infancy.
- 5 11. Use according to any of claims 1 to 10 in which the medicament comprises (1-3) IGF-I.
 - 12. Use according to any of claims 1 to 11 in which the medicament is administered systemically.
- 13. Use according to any of claims 1 to 11 in which the medicament is administered locally.
- 14. Method for influence on glutamate-receptor-controlled cells by administration of peptide antagonists at glutamate receptors.
 - 15. Method for influence on NMDA-receptor-controlled cells by administration of peptide antagonists at NMDA receptors.
- 20 16. Method according to claim 15 for preventing NMDA-receptor mediated excitatory effects such as release of neurotransmitter or peptide as well as toxic effects resulting in cell injury or death.
- 17. Method according to any of claims 14 to 16 for influence on the function of neurons or glial cells in the central nervous system.
 - 18. Method according to any of claims 14 to 17 in which the antagonists at NMDA receptors comprises glutamic acid-terminating peptides.
 - 19. Method according to any of claim 14 to 18 in which the antagonist is chosen among (1-5)GnRH, (1-3)IGF-I, (1-37)GRF and C-peptide of insulin.
- 35 20. Method according to any of claim 14 to 19 for influencing the GnRH secretion.

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- 21. Method according to any of claims 14 to 120 for the treatment of acute or chronic disorders of the central nervous system.
- 22. Method according to any of claims 14 to 20 for the treatment of hypoxic, ischemic and metabolic brain disorders such as stroke and hypoglycaemia, traumatic, radiation-induced or inflammatory injuries to the brain and chronic degenerative states.
- 23. Method according to any of claims 14 to 21 for the treatment of children during the perinatal period and infancy.
 - 24. Method according to any of claims 14 to 22 in which a medicament is administered which comprises (1-3) IGF-I.
- 15 25. Method according to any of claims 14 to 23 in which a medicament is administered systemically.
 - 26. Method according to any of claims 14 to 23 in which a medicament is administered locally.